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Review

Increased thromboembolic risk in non-neoplastic pancreatic diseases: A review focusing on acute pancreatitis

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Abstract

Venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), is a major cause of preventable morbidity and mortality in hospitalized patients. While VTE is well-established in pancreatic malignancy, its association with non-neoplastic pancreatic conditions, particularly acute pancreatitis (AP), is less clearly defined and frequently underrecognized in clinical practice. This narrative review aims to explore and synthesize existing literature on the thromboembolic complications associated with AP, highlighting the underlying pathophysiological mechanisms, clinical implications, and current gaps in prophylactic and therapeutic strategies. We reviewed published studies from major databases up to June 2025, focusing on epidemiological trends, pathological mechanisms related to inflammation-induced thrombosis, and clinical outcomes in patients with AP complicated by VTE. The review discusses pathogenesis, epidemiology, clinical features and diagnostic challenges due to symptom overlap, current pharmacologic and non-pharmacologic management strategies, and the limited but growing real-world evidence on anticoagulation in this setting.

Keywords: Acute Pancreatitis, Venous thromboembolism, Deep vein thrombosis, Pulmonary embolism, Anticoagulation.

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Introduction

Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), is a common and potentially life-threatening condition characterized by the formation of thrombi within the venous system.[1] These thrombi most frequently develop in the deep veins of the lower extremities and may dislodge, traveling to the pulmonary vasculature to cause PE. In Western regions, approximately 1 in 12 individuals will be diagnosed with VTE in their lifetime, with approximately 20% of individuals newly diagnosed dying within 1 year; hence, it remains a significant cause of morbidity and mortality worldwide.[2] In hospitalized patients, it is considered one of the most preventable causes of death.

Acute pancreatitis (AP) is an inflammatory condition of the pancreas characterized by abdominal pain and elevated pancreatic enzyme levels.[3] It represents the leading cause of gastrointestinal-related hospital admissions globally. The clinical course of AP varies from mild, self-limiting illness to severe, necrotizing disease associated with systemic complications and high mortality rates. The revised Atlanta classification system stratifies AP into mild, moderately severe, and severe categories based on the presence and duration of organ failure and local or systemic complications.[4]

While complications such as pancreatic necrosis, systemic inflammatory response syndrome (SIRS), and infection are well-recognized in AP, vascular complications, particularly VTE, remain underappreciated[5] Venous thrombosis in pancreatitis often involves the splanchnic circulation, including the portal and splenic veins.[6] However, extra splanchnic involvement, such as lower limb DVT and PE, though rare, is increasingly being recognized.(6)The pro-inflammatory and pro-thrombotic state in severe pancreatitis can predispose to VTE through mechanisms such as endothelial injury, coagulation cascade activation, and impaired fibrinolysis.[5]

Recent data suggest that hospitalized patients with AP are at increased risk of developing VTE, particularly in severe forms involving necrosis or infected collections. Patients who develop VTE during an episode of pancreatitis often experience longer hospital stays, higher healthcare costs, and increased mortality compared to those without thromboembolic complications.[7] Despite this, the incidence of VTE in AP remains poorly defined, and guidelines for screening and prophylaxis are not well established.

Although there is a well-documented association between pancreatic malignancy and thromboembolism, far fewer studies have examined the relationship between non-malignant pancreatitis (acute or chronic) and systemic VTE.

Given the potential severity and preventability of VTE in the context of pancreatitis, this narrative review aims to explore the current understanding of epidemiology, pathophysiological mechanisms, and management of VTE in acute pancreatitis. It also highlights the need for greater clinical awareness, early diagnostic strategies, and consideration of prophylactic measures in this high-risk population

Methodology

This study systematically gathered and analysed information on the pathophysiology, epidemiology and incidence, risk factors, clinical presentations, diagnostic challenges and management of VTE in AP. The methodology involved a comprehensive review of reputable sources, including scientific literature, peer-reviewed journals, and authoritative medical websites. The following steps were undertaken:

Literature Search

We conducted a thorough literature search for published manuscripts on Acute pancreatitis and venous thromboembolism in PubMed, Research gate, Google Scholar and Core databases and employed the following search terms: "venous thromboembolism", "acute pancreatitis", "pancreatitis and venous thromboembolism", "pancreatitis and thrombosis", "pancreatitis and pulmonary embolism", "pancreatitis management". Qualitative and quantitative data were extracted by interpreting each paper in cycles to avoid missing potentially valuable data. Articles consulted were not limited to studies conducted within Nigeria and the African continent, and all the databases were searched from inception to June 2025.

Inclusion and Exclusion Criteria

The search results were screened based on predefined inclusion and exclusion criterion. Only articles written in English and with full text availability were considered. Studies on human and animal subjects, clinical trials, and reviews providing comprehensive insights into VTE in AP were included. Articles in other languages or without full texts were excluded. Also, Studies on chronic pancreatitis and other forms of thromboembolism other than venous thromboembolism were excluded.

Data Extraction

Pertinent data and information were extracted from the selected articles. This included details on the pathophysiology of VTE in AP, their epidemiology and incidence, clinical features, and management. Key findings, statistics, and clinical recommendations were recorded.

Data Analysis

The extracted data was analysed and organised thematically. Similarities and patterns in the findings were identified, and key concepts related to VTE in AP were synthesised. Data were categorised into subtopics, such as pathophysiology, clinical features, epidemiology, clinical presentation and management, for a coherent presentation. All articles had their CSV (Comma Separated Value) or RIS (Research Information System) files imported into the Rayyan software and duplicates were removed. Further screening was conducted on Excel Spreadsheets (Version 2401) by two independent reviewers, O.K.E and C.B.U.

Reporting Guidelines

This narrative review was conducted and reported in accordance with the SANRA (Scale for the Assessment of Narrative Review Articles) guidelines. The SANRA checklist was used to ensure clarity, scientific rigor, appropriate referencing, and transparent presentation of findings.

Ethical Considerations

This narrative review is based entirely on previously published literature and does not involve any new studies with human participants or animals conducted by the authors. Therefore, ethical approval and informed consent were not required.

Citation and Referencing

All sources used in the study were adequately cited and referenced. The references were formatted according to the appropriate citation style (Vancouver) to ensure accuracy and consistency.

Manuscript Composition

The findings and insights from the analysis were synthesised and used to construct the study sections. By employing this methodology, this study ensured a rigorous and systematic approach to gathering and analysing relevant information on VTE in AP. The utilisation of reputable sources and adherence to inclusion and exclusion criteria enhanced the validity and reliability of the findings.

Pathophysiology of Venous Thromboembolism in acute Pancreatitis

The pathogenesis of venous thromboembolism (VTE) in acute pancreatitis is multifactorial and aligns with Virchow's Triad: venous stasis, endothelial injury, and hypercoagulability.[5] These mechanisms are amplified by the systemic inflammatory environment characteristic of pancreatitis, contributing to thrombus formation in both splanchnic and extra-splanchnic vasculature.

Endothelial Injury and Inflammatory Activation

Acute pancreatitis (AP) initiates a potent inflammatory cascade marked by the release of cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1, IL-6, and IL-10.[5] These cytokines promote endothelial dysfunction, increase capillary permeability, and impair the natural anticoagulant mechanisms. The injured endothelium becomes a prothrombotic surface, facilitating platelet aggregation and clot formation.

Pancreatic enzymes, particularly elastase, are implicated in vascular injury.[3] In some cases, pancreatic cysts connected to the pancreatic duct can rupture into adjacent vasculature, releasing proteolytic enzymes that directly damage vascular walls.[3]This enzyme-mediated vasculitis contributes to thrombus formation, especially in the pulmonary vasculature.

Additionally, nitric oxide metabolism is disrupted in AP. Studies suggest elevated plasma nitrate/nitrite levels impair endothelial-dependent vasodilation, while sensitivity to vasoconstrictors like phenylephrine is reduced, further disturbing vascular tone and promoting thrombosis in both mesenteric and pulmonary arteries.[3]

Hypercoagulability

Pancreatitis induces a hypercoagulable state through multiple pathways. Hepatic dysfunction secondary to systemic inflammation may affect the synthesis of coagulation factors. Concurrently, elevated levels of trypsin increase fibrinogen and factor VIII concentrations, shifting the hemostatic balance toward thrombosis.[3]In severe cases, cachexia and hypovolemia, driven by vomiting, third-space fluid losses, and inadequate oral intake, may lead to hemoconcentration and further enhance risk of VTE.[5]

Systemic inflammatory response syndrome (SIRS), common in severe AP, amplifies this hypercoagulable state by reducing the activity of natural anticoagulants (e.g., antithrombin III, protein C, and protein S) and impairing fibrinolytic pathways.[5] This ultimately fosters widespread microvascular thrombosis and larger venous thrombi.

Venous Stasis and Hemodynamic Alterations

Immobility, a common feature in hospitalized patients with pancreatitis, significantly contributes to venous stasis, particularly in the lower extremities.[3] Moreover, mesenteric ischemia and reduced vascular responsiveness during acute inflammation compromise blood flow in the splanchnic circulation, increasing the risk of local thrombosis.

Epidemiology and Incidence

Acute pancreatitis (AP) is a prevalent gastrointestinal disorder, with incidence rates ranging from 5 to 35 cases per 100,000 individuals globally.[8]

Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), is a significant complication in pancreatitis. In a study by Karsten Keller et al carried out in Germany, it was noted that the incidence of VTE in hospitalized AP patients was approximately 1.8%, equating to 1,764.8 cases per 100,000 hospitalizations.[9] This rate is notably higher than the general hospitalized population, where Venous Thromboembolism incidence is around 1.3% for DVT and 0.4% for Pulmonary Embolism.[10]

The prevalence of Venous Thromboembolism in Acute Pancreatitis patients varies based on disease severity and patient demographics. A study by Alexandria J Robbins et al analysing 4,613 adult patients admitted from 2011 to 2018 with Acute Pancreatitis patients found that 6.5% developed Venous Thromboembolism within the first-year post-diagnosis. These patients were more likely to be older males with underlying coagulopathies. Venous Thromboembolism occurrence was associated with increased mortality (27% vs 13%), longer hospital stays (16 vs. 5.5 days), and higher readmission rates.[10]

In necrotizing pancreatitis, the risk is even more pronounced. A retrospective review of 545 patients with necrotizing pancreatitis reported Venous Thromboembolism incidence of 57%, with 50% experiencing splanchnic vein thrombosis (SVT), 16% with extremity deep venous thrombosis, and 6% with pulmonary embolism. Multiple site venous thromboembolism was observed in 22% of cases.[11]

The severity of pancreatitis significantly influences venous thromboembolism risk. Patients with necrotizing pancreatitis exhibit higher venous thromboembolism rates, with factors such as infected necrosis, organ failure, and respiratory complications contributing to increased risk.[11]

In a prospective study of 102 acute pancreatitis patients, patients with extremity deep venous thrombosis had higher Bedside Index for Severity in Acute Pancreatitis (BISAP) scores and mortality rates. The Well's score demonstrated high sensitivity (80%) and specificity (96.9%) in predicting extremity deep venous thrombosis in these patients.[12]

This elevated risk underscores the need for vigilant monitoring and potential prophylactic measures in pancreatitis patients, particularly those with severe disease manifestations.

Risk Factors

Severity of Inflammation Acute necrotizing pancreatitis (ANP) is one of the strongest predictors of VTE.[13]A large database study by Ahmad D et al demonstrated that Venous Thromboembolism occurs in 7.1% of Acute Necrotizing Pancreatitis cases versus 2.8% in non-necrotizing pancreatitis (adjusted OR 2.78; 95% CI 2.73–2.84).[13] A single-centre cohort study by Gaurav et al also reported a 12.5% VTE incidence in Acute Necrotizing Pancreatitis, with infected necrosis being a strong predictor (peri-pancreatic necrosis OR 7.61; infected necrosis OR 2.26).[14] This may be because severe inflammation triggers cytokine-driven coagulation activation, endothelial injury, and venous stasis key elements of Virchow's triad .

Prolonged Immobility or Intensive Care Unit stay

Immobilization in hospitalized pancreatitis patients (≥2 weeks) independently raises Venous Thromboembolism risk (Acute Necrotizing Pancreatitis study: LOS ≥14 days OR 4.08; CI 1.75–9.50).[14] ICU admission, mechanical ventilation, sedation, and vasopressor use are common in severe pancreatitis and these are established risk factors for thrombosis. Prolonged bedridden state also decreases venous return and fosters stasis, further promoting thrombosis.

Central Venous Catheter Placement

Central Venous Catheter placement which is frequently required and used for total Parenteral nutrition in pancreatitis management, significantly increases the risk for Venous Thromboembolism. In an inpatient cohort study by Guru Trikudanathan et al, Central Venous Catheterization was associated with a threefold increased odds of VTE (OR 3.01), among the strongest predictors.[15] Literature by Minet C et al indicates that catheter-related thrombosis occurs in 2–69% depending on site, duration, and patient factors.[16]

Underlying Hypercoagulable States (Genetic or Acquired)

Genetic thrombophilias such as Factor V Leiden, antithrombin deficiency, protein C/S deficiency, and non-O blood type increase VTE risk by 2 to10 fold.[17] Acquired thrombophilic conditions commonly accompany pancreatitis, including: Elevated fibrinogen, Factor VIII, Inflammatory cytokines (IL-6, TNF-α) upregulating coagulation, Hypertrypsinemia and hepatic dysfunction impacting anticoagulant production.[18] Patients with coagulopathy features in pancreatitis studies had significantly higher VTE rates and worse outcomes.[10]

Clinical Presentation and Diagnostic Challenges

Dyspnea and chest discomfort are among the most frequent signs of pulmonary embolism (PE) in patients with acute pancreatitis.[19]

Lower limb leg swelling, redness, or pain from deep vein thrombosis (DVT) overlaps with leg edema secondary to hypoproteinemia or fluid shifts, common in pancreatitis, making recognition more difficult. A case report by Xiu-Ling Fu et al highlighted that dyspnea in acute pancreatitis can mask underlying Pulmonary embolism without overt clinical clues.[19].

Some of the diagnostic challenges include masking of VTE Signs by Pancreatitis Symptoms. Pancreatitis often presents with abdominal pain, nausea, tachycardia, and fever, which can mimic or conceal Pulmonary Embolism or Deep venous thrombosis symptoms. Systemic inflammation and fluid shifts may cause tachypnea, hypoxia, and leg edema, already present without thrombotic events, confusing the clinical picture. As such, persistent dyspnea or unilateral leg swelling in pancreatitis patients should raise suspicion for concurrent Venous Thromboembolism even when background symptoms are present.

Prolonged hospital stays.

VTE in pancreatitis correlates with increased length of stay, mortality, and morbidity; a study by Asuncion et al reported mortality that was more than twofold higher in hospitalized pancreatitis patients with VTE (7.5% vs. 2.9%).[20] Awareness of VTE risk, especially in severe or necrotizing pancreatitis, long hospital stays, immobilization, or ICU admission, should prompt screening and close monitoring.

Role of Biomarkers (D-dimer)

D-dimer is highly sensitive (~98%) for venous thromboembolism but has low specificity.[21] Levels are often elevated in severe pancreatitis due to systemic inflammation; a recent study done in Thrissur from October 2022 to March 2024 among 60 patients reported D-dimer levels >2528 ng/L to be strongly associated with severe acute pancreatitis (AUC 0.87).[22] Because of poor specificity in pancreatitis, D-dimer is best used to rule out VTE in low pretest probability patients, not to confirm it.[23]

Imaging Modalities

CT Pulmonary Angiography (CTPA)

CTPA is the diagnostic gold standard for Pulmonary Embolism, recommended when pre-test probability is intermediate or high. In pancreatitis, the performance remains reliable, though contrast may confound peripancreatic fluid or abscess regions. It may complicate interpretation in inflamed tissues.

Doppler Ultrasound

Lower-extremity Doppler ultrasound detects proximal DVT via vessel compressibility. This is usually the first line for evaluation of Deep venous thrombosis. Identifying DVT may exclude the need for CTPA in patients without pulmonary symptoms.

Other modalities include Contrast-enhanced CT, MRI venography, 99mTc venography, and lung perfusion scans can detect splanchnic thrombosis or PE where standard imaging is limited.[24]

Management and Prophylaxis

Currently, there is no established consensus on the optimal management of venous thromboembolism (VTE) in patients with acute pancreatitis (AP). The literature remains divided: some studies support the routine use of anticoagulation in AP, especially in cases with a higher incidence of VTE particularly splanchnic vein thrombosis (SVT) while others advise caution due to the associated hemorrhagic risks and a lack of evidence demonstrating significant mortality or morbidity benefit from anticoagulation in this setting.[25]

Early Recognition and Initial Management

Prompt identification and investigation of thromboembolic events in AP are critical, as timely diagnosis and appropriate intervention can significantly reduce mortality. Intravenous unfractionated heparin has shown effectiveness in early treatment. In select cases, inferior vena cava filters have been utilized to prevent pulmonary embolism (PE), although evidence for their routine use remains limited. Pulmonary embolism, although rare in AP, poses a significant risk and requires swift and aggressive treatment once identified.[26,27]

Prophylactic Strategies

Preventing VTE in patients with necrotizing pancreatitis (NP), particularly those with severe disease and prolonged hospitalization, necessitates a shift toward more aggressive prophylactic and screening protocols.[28] High-risk individuals defined by organ failure, infected necrosis, a personal or family history of VTE, or prolonged immobility may benefit from enhanced surveillance strategies such as weekly four-limb Doppler ultrasound screening. The development of a validated VTE risk scoring system specific to pancreatitis could aid in stratifying patients and guiding prophylactic decisions.[28]

Pharmacologic Prophylaxis

Pharmacological prophylaxis with anticoagulants such as low-molecular-weight heparin (LMWH), unfractionated heparin, or fondaparinux has demonstrated safety and efficacy in reducing VTE with reduction in relative risk of about 40-70%. The American College of Chest Physicians (CHEST) guidelines recommend these agents in non-surgical hospitalized patients unless contraindicated.[29-31] However, prophylactic anticoagulation in pancreatitis requires careful consideration due to the potential for hemorrhagic complications, particularly in patients undergoing invasive procedures or those with pancreatic necrosis.[32]

Anti-factor Xa level monitoring and dose adjustments may be warranted in select high-risk individuals, drawing from protocols established in trauma populations.[28] Lacking a standard of care in the current clinical practice, the decision to use pharmacological prophylaxis is made on a case-by-case basis.

Further research is needed to determine the specific recommendations for VTE prophylaxis in patients with pancreatitis deemed high risk.[32]

Real-World Evidence and Case Insights

A Case report by Alomar et al illustrated that even standard-dose enoxaparin (40 mg daily), considered adequate prophylaxis, may fail in the proinflammatory context of severe pancreatitis. The patient in this report had long intensive care units (ICUs) stay with decreased mobility and had complications such as pancreatic abscesses.[33]This highlights the potential for an overwhelmed anticoagulant response due acute inflammatory state.

Inflammatory mediators (e.g., TNF-α, IL-1, IL-6) released during pancreatitis disrupt endothelial integrity and activate the coagulation cascade, like mechanisms observed in malignancy-associated thrombosis. Some evidence suggests that direct oral anticoagulants (DOACs), such as rivaroxaban, may offer efficacy comparable to LMWH in this context. However, standardized dosing protocols in acute pancreatitis are lacking, and current escalation strategies in dose of enoxaparin are limited to patients with high body mass index (>40), leaving a critical gap in management for non-obese patients experiencing this proinflammatory condition.[33,34]

Need for Further Research

Despite the recognition that acute necrotizing pancreatitis significantly increases the risk of VTE, particularly within the first 30 days of onset, there remains insufficient evidence to clearly define who should receive anticoagulation and which agents or doses are most effective. Prospective studies are urgently needed to evaluate the efficacy of intensified prophylactic strategies, including higher anticoagulant dosing, in high-risk AP and NP populations. Furthermore, investigations should explore the safety, timing, and duration of anticoagulation both during hospitalization and in the early post-discharge period.

Conclusion

Acute pancreatitis contributes to a pro-thrombotic state via mechanisms involving endothelial injury, systemic inflammation, and venous stasis. The risk is particularly pronounced in patients with necrotizing disease, prolonged hospitalization, or additional risk factors such as central venous catheters or immobility. Despite emerging data, standardized guidelines for VTE prophylaxis and treatment in pancreatitis are lacking. This review underscores the urgent need for greater clinical awareness and further research to inform evidence-based management of VTE in non-neoplastic pancreatic disease.

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